APPENDIX A

FOCUSED REVIEW

Neurogenic Bowel Dysfunction After Spinal Cord Injury: Clinical Evaluation and Rehabilitative Management

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ABSTRACT. Stiens SA, Biener Bergman S, Goetz LL. Neurogenic bowel dysfunction after spinal cord injury: clinical evaluation and rehabilitative management (Focused review). Arch Phys Med Rehabil 1997;78:S-86–S-102.

• Neurogenic bowel dysfunction (NBD) is one of many impairments that result from spinal cord injury (SCI). The experience of persons with SCI reveals that the risk and occurrence of fecal incontinence and difficulty with evacuation are particularly significant life-limiting problems. This review relates the anatomy and physiology of colon function to the specific pathophysiology that detracts from the quality of life of persons after SCI. There are two patterns of NBD after SCI: the upper motor neuron bowel, which results from a spinal cord lesion above the sacral level, and the lower motor neuron bowel, which results from a lesion to the sacral spinal cord, roots, or peripheral nerve innervation of the colon. Rehabilitation evaluation consists of a comprehensive history and examination to define impairments, disabilities, and handicaps pertinent to NBD. Rehabilitation goals include continence of stool, simple willful independent defecation, and prevention of gastrointestinal complications. Intervention consists of derivation and implementation of an individualized person-centered *bowel program*, which may include diet, oral/rectal medications, equipment, and scheduling of bowel care. *Bowel care* is a procedure devised to initiate defecation and accomplish fecal evacuation. *Digital-rectal stimulation* is a technique utilized during bowel care to open the anal sphincter and facilitate reflex peristalsis. Recent advances in rehabilitation practices, equipment, pharmacology, and surgery have offered patients new bowel program alternatives. Interdisciplinary development of solutions for problems of NBD are evolving rapidly.

INTRODUCTION: THE EFFECTS OF COLONIC IMPAIRMENT ON THE PERSON

Spinal cord injury (SCI) produces a wave of repercussions that affect many organ systems and subsequently the life activities of the affected person.[1,2] SCI has obvious effects on mobility which limit a person's ability to traverse and modify the surrounding physical environment. What is less appreciated by the general population is the effect of SCI on a person's ability to control the elimination of stool and urine. To compound these problems, impairments of sphincter control and gross mobility interact to limit alternatives for self-management of the resultant incontinence.[3] More than one-third of surveyed subjects with SCI rate bowel and bladder dysfunction as having the most significant effect on their lives after injury,[4] and many rank neurogenic bowel dysfunction one of their major life-limiting problems.[5-8] In the Stockholm SCI study, 41% of subjects rated bowel dysfunction as a moderate to severe life-limiting problem.[9] Much of this opinion may result from the limited rehabilitation interventions that are available to reduce the functional effects and life impact of three impairments: constipation, incontinence, and immobility.

Disabilities that limit willful continence, voluntary defecation, and perineal hygiene are difficult to fully remedy. The options for independent self-management of neurogenic bowel dysfunction are limited, and attendant care is costly.[10-15] Boss and associates[16] studied the self-care capabilities of a cohort of outpatients with SCI. Not surprisingly, bowel management was identified as an area of least competence. They recommended reevaluation of agencies' bowel management self-care teaching protocols after observing difficulties in acquiring bowel care competence and independence by many clients with SCI. Indeed, for many, colonic dysfunction is a source of continuing inconvenience, frustration, and expense.[17] In the interim period before personal care attendants needed by the individual are funded and hired, visiting nurse or home care agencies may be brought in to assist with personal care. In many areas, home health aides are not licensed to perform bowel care, which is considered invasive. Thus, it must be performed by an RN or LPN. The cost of this level of expertise is high, and staffing difficulties drive a policy of attempting to teach family members to perform bowel care. However, having a family member perform such intimate tasks can be emotionally charged and may have negative effects on family relationships.

The effects of the neurogenic bowel on the quality of life after SCI are, unquestionably, significant. Coping with potential incontinence requires a combination of time, expense, and attendant support.[18] Fear of bowel accidents is a commonly stated reason why persons with SCI do not engage in activities outside of the home or travel away from home, and so the impact of poor bowel management actually extends far beyond impaired intestinal motility.[19] Yet despite

the magnitude of the problem, bowel dysfunction after SCI has yet to be fully understood or effectively treated.

The physical symptoms associated with hindgut dysfunction can further detract from life experience after SCI. Complaints relative to the gastrointestinal system are frequent after SCI.[5,7] Stone and associates[5] observed that 29% of persons with complete SCI reported bowel-related problems. Symptom frequency increased in subjects who had been spinal cord injured for more than 5 years.

Unfortunately, these reported symptoms may be just the tip of the iceberg, since much of the pathology associated with the intestinal tract after SCI remains occult. Clinical recognition of acute intra-abdominal pathology is hampered by diminished visceral sensitivity. In a retrospective review of gastrointestinal complications after SCI, approximately one-half of the recognized complications occurred during the first month after injury.[20,21] Consequently, the clinician is often confronted with a complex rubric of nonspecific symptoms, which may or may not be related to the large variety of unheralded disorders that may be harbored within a patient's insensate gut. Judicious practice requires close surveillance for signs of complications and a thorough evaluation of symptoms.

The comprehensive practice of rehabilitation medicine must address the resultant disablement experienced by the person with SCI and concurrently prevent gastrointestinal and other medical complications. This review will begin with illustrations of pertinent normal anatomy and physiology required for understanding the pathophysiology of the neurogenic bowel after SCI. Next, the variety of impairments of colonic function due to SCI will be described and contrasted with normal function. Then, the effect of these impairments on the person's life as they interact to limit activity will be explored. Finally, techniques, equipment, and medications for rehabilitation management will be outlined. Discussions will focus on surveillance, prevention, and practical solutions to limit further disability.

HINDGUT ANATOMY: ORGAN STRUCTURE AND NERVOUS INNERVATION

The human large intestine is a closed, compliant sac, averaging 1.5m in length, that is bounded by the ileocecal sphincter at one end and the anal sphincter at the other. It is divided into five parts: appendix, cecum, colon, rectum, and the anal canal.[22] The colon wall consists of an inner layer of circular smooth muscle and a thin outer layer of longitudinal smooth muscle that is gathered into three thick cords called the *taenia coli*. The left transverse colon, descending colon, and rectum are derived from the embryonic hindgut and connect to the skin surface through the anus.

At the caudal rectum, the continuous inner smooth muscle layer of the colonic wall thickens to produce the internal anal sphincter (IAS). The IAS surrounds the anal canal at the distal rectum, and is the major contributor to the resting pressure of the closed anal canal.

Located just inside the anus, the external anal sphincter (EAS) is a circumferential band of striated muscle that is

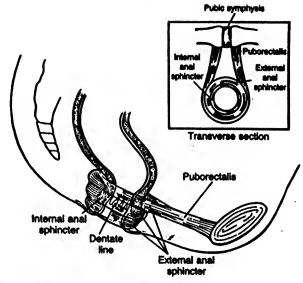

Fig 1. Sagittal view of the rectum, anal canal, and surrounding muscles. The puborectalis muscle forms a sling posteriorly around the rectum at the anorectal junction. The external anal sphincter (skeletal muscle) surrounds the anal canal and is closely associated with the puborectalis muscle. The internal anal sphincter (smooth muscle) lies within the ring of external sphincter muscles and is a continuation of the inner circular layer of the smooth muscles of the rectal wall. (from Madoff RD, Williams JG, Caushaj PF. Fecal Incontinence, N Engl J Med 1992;326:1002-7, used with permission)

continuous with the pelvic floor. The puborectalis muscle loops around the distal rectum and maintains the anorectal angle by tethering the rectum toward the pubis, thus maintaining continence (fig. 1). The levator ani muscles arise from the posterior pubic bone and attach to the distal sacrum and coccyx. This set of muscles converges to form a funnel arising from the sides of the pelvis. The urethra, vagina, and anorectum pass through this funnel to enter the perineum.

The anal sphincter mechanism (EAS, IAS, and the puborectalis) acts as a unit to maintain fecal continence. Continence in the resting state is maintained by the tonic activity of the IAS, accounting for 80% of resting sphincter pressure.[23] Rectal distention and puborectalis stretch produce the urge to defecate.[24] Reflex contraction of the EAS and puborectalis prevent incontinence with cough or Valsalva forces.

The hindgut and pelvic floor receive parasympathetic, sympathetic, and somatic innervation. Stool propulsion and storage are orchestrated by a bureaucracy of nerve cells linked from the colonic mucosa to the brain (fig. 2). This nervous network can be described starting at the colonic wall and proceeding to the spinal cord and brain.

At the level of the gut, the enteric nervous system, or "brain of the gut," is made up of 100 million neurons, approximating the nerve cell numbers of the entire spinal cord.[25] This intrinsic nervous system of the intestine includes Auerbach's plexus (intramuscular myenteric), which is situated between the longitudinal and the circular muscle layers of the intestine. Auerbach's plexus contains unmyelinated fibers and

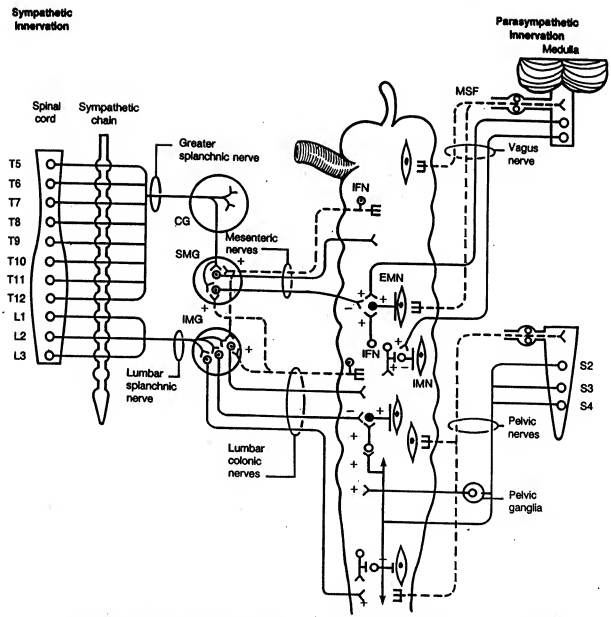

Fig 2. Autonomic control of large intestine motility. The colon is vertically diagrammed at the center of the figure, with the cecum superior and the rectum inferior. Preganglionic neurons in the thoracolumbar segments pass through the sympathetic chain to synapse with postganglionic neurons on the celiac ganglion (CG), superior mesenteric ganglion (SMG), and inferior mesenteric ganglion (IMG). Postganglionic neurons on the SMG and IMG synapse with myenteric neurons and smooth muscles cells in the proximal and distal colon, respectively. Intestinofugal mechanosensitive sensory neurons (IFN) in the myenteric plexus synapse with postganglionic sympathetic neurons in the SMG and IMG. Vagal nerves to the proximal colon and pelvic nerves to the distal colon, rectum, and internal anal sphincter (IAS) contain both efferent and mechanosensory fibers. The preganglionic parasympathetic efferent fibers make excitatory synapses with myenteric neurons. Preganglionic parasympathetic fibers in the pelvic nerves (also called pelvic splanchnic nerves and nervi erigentes) also synapse with postganglionic neurons in pelvic ganglions. (EMN, excitatory myenteric motor neurons: IMN, inhibitory myenteric motor neurons.) (from Smith T, Sanders KM. Motility of the large intestine. In:Yamada T, Alpers DH, Chung O, Powel DW, Silverstein FE, editors. Textbook of gastroenterology. Lippincott, 1995, used with permission.)

postganglionic parasympathetic cell bodies that coordinate peristalsis. Secondly, Meissner's plexus in the submucosa relays local sensory and motor responses to Auerbach's plexus, prevertebral ganglia, and the spinal cord.

Enteric nervous control of the colonic wall is modulated through central connections from parasympathetic and sympathetic nerves (fig. 3). Efferent projections are parasympathetic and sympathetic. Preganglionic cholinergic parasym-

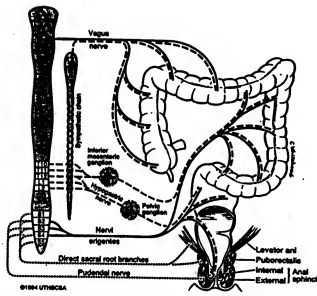

Fig 3. Autonomic and somatic innervation of the colon, anal sphincters, and pelvic floor. Spinal cord segments and nerve branches are illustrated. Dashed lines represent sympathetic pathways with prevertebral ganglia. Solid black lines depict parasympathetic pathways that synapse with ganglia in the myenteric nervous system within the colon wall. The vagus originates from the medulla and the nervi erigentes (also called the pelvic splanchnic nerve) come from the conus. Dotted lines represent mixed nerves supplying somatic musculature of the external anal sphincter and the pelvic floor. (from Cardenas DD, Mayo M, Kling JC. Urinary tract and bowel management in the rehabilitation setting. In: Braddom R, editor. Physical medicine and rehabilitation. Philadelphia: Saunders, 1996, used with permission.)

pathetic neurons produce excitatory effects through nicotinic and some muscarinic receptors.

Postganglionic sympathetic efferents are adrenergic neurons directed at four primary targets: secretomotor neurons (containing vasointestinal peptide), presynaptic cholinergic nerve terminals, submucosal blood vessels, and gastrointestinal sphincters.[25] Afferent sensory fibers are carried by the vagal, pelvic, and hypogastric nerves and respond to a wide variety of mechanical and chemical stimuli.

The colon's extrinsic nerve supply includes parasympathetic, sympathetic, and somatic nerves. The vagus ("vagabond" or "wanderer") nerve wanders from the brainstem to provide parasympathetic influence for the gut from the esophagus all the way to the splenic flexure of the colon. The pelvic nerve (also called nervi erigentes or inferior splanchnic nerves) carries pelvic parasympathetic fibers from S2-S4 conal spinal cord levels and joins the hypogastric artery to enter the descending colon and rectum. The pelvic nerve branches travel proximally within the colon to innervate the entire colon.[26] The mesenteric (T5-T12) and the hypogastric (lumbar colonic T12-L3) nerves bring sympathetic innervation to the colon. The EAS is supplied by the somatic pudendal nerve (S2-S4), which innervates the pelvic floor.

Many pathways from various brain structures through the spinal cord to the conus medullaris have been demonstrated with regional brain stimulation. None has been found to be essential for colonic function. Contractions of the colon can be divided into individual phasic, organized group (colonic migrating or nonmigrating), and special propulsive (giant migrating) contractions.[26]

HINDGUT PHYSIOLOGY: STORAGE, PROPULSION, AND DEFECATION

Although the colon is not essential for life, many important colonic functions have been identified. The colon serves as a stool reservoir, provides for growth of symbiotic bacteria,[27] secretes mucus for feces lubrication, and propels stool toward the anus. It resorbs electrolytes, short-chain fatty acids, bacterial metabolites, and fluids from the feces (up to 30L/day). In the proximal colon, the predominant pattern of contraction is rhythmic antiperistalsis produced by mixing and kneading contraction of the circular muscle layers. In the distal colon, rhythmic propulsive peristalsis predominates.[22] Normal colonic transport takes 12 to 30 hours from the ileocecal valve to the rectum.[28]

Small and large intestinal movement is largely autonomous, with some spinal cord but minimal brain influence. The colon generates intrinsic rhythmic slow waves that originate from the submucosal plexus and occur sequentially at adjacent points moving along the colonic axis. The location of origin of slow waves along the colon varies like a wandering pacemaker. Slow waves may travel toward or away from the ileocecal valve in the right colon but are consistently directed to the anus in the left colon.[22]

Control of colonic motility is exercised via three primary mechanisms: myogenic, chemical, and neurogenic.[29] The physiology of myogenic motor control is not yet fully understood. Myogenic transmission of signals occurs between enteric smooth muscle cells that are interconnected with gap junctions, producing a syncytium of smooth muscle. These gap junctions provide electrical coupling and allow oscillations of muscle contraction. Most intestinal muscle displays autorhythmicity. The basal electrical rhythm of the colon is characterized by slow waves with a variable frequency of 2 to 13 cycles per minute, without a frequency gradient along the viscus. Highly variable and intermittent spike potential bursts may oscillate at between 1 and 60 cycles per minute and are associated with measurable colonic wall contractions.[22]

Chemical control acts by modulation of colonic contractile activity with substances liberated from nerve endings, varicosities, and endocrine-paracrine cells. These chemicals may exert influence at the central nervous system, autonomic nervous system, enteric nervous system, or muscle cells to promote or inhibit contractions. These substances include: amines, peptides, acetylcholine, and fatty acid derivatives (prostaglandins and sex steroids).[26,29]

The most local neurogenic mechanism of colonic control comes through the enteric nervous system, which coordinates all segmental motility and some propagated movement. Conus-mediated reflexes produce holocolonic propulsion of

stool via pelvic nerve pathways.[26] Moving from the colonic wall to the spinal cord and brain, reflexes can be segregated into the following categories: (1) enteric, (2) prevertebral and vertebral, (3) vagal, and (4) pelvic.[26]

Operating independently within the colonic wall, enteric reflexes do not require extrinsic colonic innervation. In 1899, two British physiologists, W. M. Bayliss and E. H. Starling, reported that the intestines, even when removed from the body, have an inherent tendency to produce peristalsis toward the anus.[30] This colo-colonic intramural reflex has become known as "the law of the intestine." Whenever the intestinal wall is stretched or dilated, the nerves in the myenteric plexus cause the muscles above the dilation to constrict and those below the dilation to relax, propelling the contents caudally (fig. 2). This intramural wiring provides the small and large[31] intestines with directional specificity and provides descending inhibition that facilitates bolus transfer.

Prevertebral reflexes traverse the spinal cord to pathways through the prevertebral sympathetic ganglia, which are located outside the spinal cord. Vertebral reflexes run through the spinal cord. These circuits have been hypothesized to be primarily inhibitory and to aid in the storage function of the colon.[32,33]

The *gastrocolonic response* or "gastrocolic reflex" is initiated by feeding, which induces increased propulsive small intestine and colonic motility mediated by cholinergic motor neurons. The terminology is inaccurate on three points. The stimulus likely does not come from the stomach, the response is not exclusively confined to the colon, and the mechanism is not only through a neural reflex.[34] The exclusion of the esophagus and the stomach from the food stream does not prevent the response.[22] This postprandial response has been divided into early (within minutes) and late (lasting for up to hours) components.[35,36] The mechanism for activation of this cholinergic stimulation has yet to be conclusively defined. Proposals include central vagal mediation, intrinsic neural pathways within the colon, and humoral mediation via gastrin,[37] motilin,[38] or cholecystokinin. The response may be facilitated by a fatty or proteinaceous meal or blunted by atropine. Some investigators have reported that the gastrocolic response is less robust or absent after SCI.[39,40] A preliminary study of postprandial gut hormone response has suggested that neural mechanisms for motilin release may be impaired after SCI.[38]

Vagal reflexes include vago-vagal pathways to and from the ascending and transverse colon. Vagal stimulation increases rhythmic segmental contractions limited to the proximal colon. The movements produced are only partially blocked by atropine, which suggests the existence of both cholinergic and noncholinergic vagal efferents and intracolonic neural circuits.[26]

Pelvic reflexes are excitatory and are relayed by the pelvic nerve from the colon to and from the sacral spinal cord segments within the conus medullaris. Stimulation of the parasympathetic pelvic splanchnic nerve can produce explosive motility of the entire colon.[26] Spinal cord-mediated reflexes via the pelvic nerve are initiated from enteric circuits in response to colonic dilation, and serve to reinforce colonic-initiated propulsive activity.[26] The recto-colic reflex is a pelvic nerve-mediated pathway that produces propulsive colonic peristalsis in response to chemical or mechanical stimulation of the rectum and anal canal. Fecal continence is maintained by a closed internal sphincter. Internal sphincter closure is maintained by tonic excitatory sympathetic (L1-2) discharges. Internal sphincter tone is inhibited with rectal dilation by stool (rectal-anal inhibitory reflex) or digital stimulation. Those experienced in bowel care are frequently able to palpate an increase in internal sphincter tone after defecation, which is a clinical sign that facilitated defecation has been completed.

It is speculated that the central nervous system has only a minimal and intermittent effect on human colonic motility.[26,41,42] Mammalian studies that have included stimulation of various regions of the telencephalon, hypothalamus, and mesencephalon have documented colonic facilitation and inhibition. This modulation can occur in extreme emotional states or in the conscious act of defecation. Central voluntary control of abdominal musculature and the EAS allows willfull defecation at times of increased colonic motility.

In the neurally intact state, the process of voluntary defecation begins with spontaneous involuntary peristaltic advancement of stool into the rectum (table 1).[26] This advancement frequently occurs in response to a giant migratory contraction, often following physical activity or a meal. Giant migratory contractions occur once to twice per day and aborally propagate stool up to one-third the colonic length (mass movement). As stool is advanced against the internal sphincter, an intrinsic colonic reflex permits internal sphincter relaxation (recto-anal inhibitory reflex). The perception of rectal and puborectalis distention produces the urge to defecate.[24] Stool is momentarily retained by the conal-mediated reflex contraction of the EAS (holding reflex) and voluntary EAS contraction.

Stool is voluntarily eliminated by relaxation of the puborectalis muscle and the EAS. This produces a straighter anorectal tunnel to guide out stool driven by peristalsis and intra-abdominal pressure.

PATHOPHYSIOLOGY: HINDGUT DYSMOTILITY AND PELVIC FLOOR DYSFUNCTION

"Neurogenic bowel" is a term that relates colon dysfunction (such as constipation, incontinence, and discoordination of defecation) to lack of nervous control. Patterns of dysfunction are described in relation to neural lesions located within the brain or along the spinal cord, the peripheral nerves, and within the enteric nervous system. The effects of these lesions can be reviewed by starting at the muscle layers of the colon and working toward the spinal cord and supraspinal centers.

The enteric nervous system remains functionally intact after traumatic SCI, although the transsynaptic effects of central and peripheral lesions have been demonstrated to induce ganglion cell loss and Schwann cell proliferation within the colon wall.[43,44] Local synaptic remodeling would be expected to occur in response to spinal lesions, but the effects on enteric gut nervous control, if any, are unknown.

Table 1: Sequence of Events In Normal Defecation

Involuntary activity
1. Giant migratory contractions advance stool through the colon to the rectum.
2. Stool distends the rectum and stretches the puborectalis muscle as the internal sphincter relaxes—the rectoanal inhibitory reflex. This triggers a conscious urge to defecate.
3. External anal sphincter and puborectalis muscle contraction retains stool—the holding reflex.
Voluntary activity
1. Relaxation of the external anal sphincter and the puborectalis
2. Contraction of the levator ani, external abdominals, and diaphragm, combined with glottis closure, elevates intra-abdominal pressure and aids peristalsis in propelling stool out.

The lower motor neuron (LMN) bowel represents a pattern of colonic dysfunction that results from a lesion affecting parasympathetic cell bodies at the conus, their axons in the cauda equina, or the pelvic nerve. The LMN colon tends to be "relaxed"; no spinal cord-mediated reflex peristalsis occurs. Slow stool propulsion with segmental colonic peristalsis is coordinated by the myenteric plexus alone while water absorption continues. This situation produces an inspissated, rounder (scybalous) stool shape. The external anal sphincter is denervated, increasing the risk for incontinence. The rectal-anal inhibitory reflex is retained even in lesions of the conus medullaris or cauda equina.[43,45] The levator ani muscles lack tone and allow the sigmoid and rectum to descend into the pelvis and the perineum to bulge down, reducing the rectal angle and opening the rectal lumen.[46]

The upper motor neuron (UMN) bowel results from a lesion of the spinal cord above the conus medullaris. Lesions above T1 are associated with prolonged mouth-to-cecum transit, but paraplegics exhibit transit times that are comparable to normal.[47] Beyond the ileocecal valve, the UMN colon has been described as "spastic" because of the excessive colonic wall and anal tone observed. The striated EAS muscle, normally under voluntary control, remains tight as a result of spasticity of the pelvic floor.[48] Surface electromyography studies have demonstrated that persons with UMN SCI have a higher basal colonic activity than normals.[39,44] This could lead to overactive segmental peristalsis, underactive propulsive peristalsis, and a hyperactive holding reflex with spastic EAS constriction, producing fecal distention of the colon. This condition necessitates a mechanical[49] or chemical stimulus to trigger reflex defecation.

Early studies of colonic compliance of the UMN colon were reported in the 1940s.[50] Subsequent investigations[51] have included colometrograms in which water was infused into the rectum at a rate of 100ml/min with continuous intracolonic pressure measurement. Rapid rises in intracolonic pressures were observed in subjects with UMN SCI, exceeding 40mm Hg (normal 5-15 mm Hg) at volumes of 400 to 600ml.[40] These and other studies[49,52] have suggested a hyperreflexic response of the left colon that is analogous to that of the UMN bladder. Subsequent studies with gradual or intermittent rectal infusion have demonstrated pressure reduction with colonic compliances which have appoached normal.[53,54]

The effect of UMN SCI on colonic transport has been investigated with various techniques.[28,43,45,55] As reviewed by Wald,[56] these techniques could be useful for clinical evaluation of colonic and anorectal motility. Transit times through the colon can be calculated by counting swallowed (N = 20) markers dispersed with peristalsis and followed through the colon with serial radiographs.[57] Using this technique, investigators working with subjects with UMN SCI have demonstrated markedly delayed left colon and rectal transit times as compared with controls.[28,45] Recently, colonic transport was investigated in seven subjects with UMN SCI using indium-111 amberlite scintigraphy.[55] A single capsule containing amberlite IR-120-P cation exchange resin pellets was ingested orally. The isotope was encapsulated in gelatin and coated with methacrylate to allow release in the colon. A significantly slower emptying half-time was observed (ascending colon: 29 ± 27h for subjects with SCI, compared with 6.81 ± 3.03h for controls, $p < .01$; ascending and transverse colon: 42 ± 12h for subjects with SCI, 15.3 ± 7.16h for controls $p < .01$. These results suggest that abnormalities of colonic function after UMN SCI can involve the entire colon and may require the use of oral agents in addition to rectal medications and reflex stimulation.

To summarize, the LMN bowel syndrome produces constipation with a high risk of frequent incontinence through a lax external sphincter mechanism. The UMN bowel syndrome includes constipation with fecal retention behind a spastic anal sphincter, requiring a chemical or mechanical trigger for defecation.

REHABILITATION INTERVENTION FOR NEUROGENIC HINDGUT DYSFUNCTION: MINIMIZATION OF THE ASSOCIATED IMPAIRMENTS, DISABILITIES, AND HANDICAPS

The approach to the problems caused by neurogenic bowel is the same as for all issues that confront the patient in the rehabilitation process.[2,3] The rehabilitation database must include pertinent aspects of anatomic injury and diagnoses that relate to the colon. Impairments of colonic function must be derived from history, examination, and testing. Next, the problem is succinctly described and included on the rehabilitation problem list as a UMN or an LMN pattern of impairment. Specific difficulties such as constipation, difficulty with evacuation, and fecal incontinence must be understood and quantified. Disabilities that limit a person's ability to maintain continence and willfully defecate must be assessed within the perspective of the entire person. Next, liabilities of functional mobility, as well as assets, such as residual colonic reflex function, that can be exploited in

Table 2: Baseline Medical History for Neurogenic Bowel

Premorbid history
Daily fluid intake, diet (fiber, meal frequency, spice preferences, amounts), bowel movements (frequency, duration, difficulties), stool (consistency, color, mucus, blood), medications
Current status
Injury level, daily fluid intake, diet, medications, patient's understanding of effect of spinal cord injury on elimination, bowel care (frequency, duration, digital stimulation, frequency/technique), bowel incontinence (time of day, frequency, relationship to eating)
Lifestyle goals
Schedules for work or school, availability of assistance if needed, amount of time needed to complete bowel care regimen.

bowel care maneuvers through task modification or pharmacology, must be sought. Finally, issues of handicap need to be addressed with consideration of the person's life goals and role expectations, particularly cultural, sexual, and vocational roles. The entire process therefore requires knowledge of the individual person and derivation of person-centered goals.[2] A particular team member should coordinate this interdisciplinary intervention effort. Typically, this member is the primary nurse in close association with the physiatrist, occupational therapist, and others on the interdisciplinary rehabilitation team.

The postrehabilitation lifestyle goals and needs of the individual after the acute rehabilitation process is complete must determine the timing and content of a new bowel care schedule. The individual's demands of work or school, the duration of bowel care, and the needs of other members of the household must all be considered in scheduling. In the inpatient setting, scheduling can be especially difficult because of the time-consuming nature of bowel care, and the number of persons who require bowel care assistance.[10] Evening bowel care often allows more predictable attendance at daily therapies.

It is crucial to remember that the patient must take a leadership role in building a bowel program that incorporates a life-compatible bowel care schedule. Our job is to educate him or her about altered physiology after SCI and to empower him or her to construct a bowel care regimen that he or she can live with.

PATIENT HISTORY: WHAT IS THE PATTERN AND FIT OF BOWEL FUNCTION IN THE PERSON'S LIFE?

Appropriate management of the neurogenic bowel begins with a thorough rehabilitation history. The questions to be asked in the initial interview are summarized in table 2. Previous bowel conditions and habits must be related to current function, support resources, and life demands.

Premorbid bowel function lays the groundwork for developing a workable postinjury bowel regimen. Parameters to consider include timing of bowel movement, the frequency, volume, and consistency of bowel movements, and the amount of time needed to complete bowel care. These should be replicated as closely as possible in order to take advantage of well-established patterns. Scheduling elimination shortly after a meal takes advantage of the gastrocolic response.

The presence of any premorbid bowel disorder will also complicate a postinjury bowel regimen. Laxative dependency, diabetes, irritable bowel syndrome, or inflammatory bowel disease can prolong transit time, decrease the responsiveness of the gut to bowel care medications, and even predispose the person to life-threatening complications, such as toxic megacolon. These persons need to be very compliant with bowel programs. They need to understand the relationship between their previous problem and their post SCI bowel dysfunction.

PHYSICAL EXAMINATION: IDENTIFICATION OF IMPAIRMENTS, SURVEY FOR COMPLICATIONS, AND PATIENT EDUCATION

Suspected colonic and pelvic floor dysfunction is confirmed, and capabilities for compensation are explored. One should note the patient's general body habitus, hydration status, and level of alertness. The abdomen should be inspected for distention. Auscultation of bowel sounds precedes palpation. Spasticity of the abdominal muscles can be minimized by supporting the flexed knees with a pillow. Palpation will reveal the presence of any masses, organomegaly, or high colonic impaction.[58] The exam may not yield abdominal tenderness, rigidity, or rebound because of alteration in sensory innervation. The colon should be palpated along its course as it encircles the abdomen, starting in the right lower quadrant. The ascending colon and transverse colon have varied positions in the abdomen. The descending colon reliably follows the left lateral abdomen and becomes retroperitoneal as the colon descends toward the pelvis.

A careful rectal examination will also yield information about sphincter innervation and the amount and consistency of stool present in the rectal vault, the presence of hemorrhoids or masses, and in males the size of the prostate gland. A stool guaiac should be checked for occult blood at this time. On rectal examination, the examining finger should be held firmly against the anal verge to allow gradual relaxation of anal tone. As the sphincter opens, the examining finger should be directed toward the umbilicus in order to approach the rectal angle at the puborectalis muscle. At the puborectalis, gentle pressure toward the sacrum is applied to assess the puborectalis for tone and spasticity. Assessment should be made of the voluntary strength of the EAS, puborectalis, and bulbocavernosus. Strength should be graded, and endurance can be assessed.[59] The neurologic examination will yield information about the level of injury, completeness of the injury, and whether the lesion is of the UMN or LMN type.

The neurologic exam should include a thorough assessment of sacral reflexes, including anal tone, anocutaneous

reflex, bulbocavernosus reflex, and the internal anal sphincter reflexes. Contraction of the IAS with the stimulation of digital introduction to the rectum beyond the EAS constitutes the IAS reflex. The sphincter is closed immediately on withdrawal. This reflex is sympathetically mediated through the hypogastric plexus. The anocutaneous reflex, which consists of contraction of the EAS in response to pricking or stroking the anal skin, should be assessed in all four quadrants that surround the anal verge. This reflex is mediated by the inferior hemorrhoidal branch of the pudendal nerve (S2-S5).

The bulbocavernosus reflex is elicited by pricking or pinching the dorsal glans penis (S3) or pressing the clitoris and palpating for bulbocavernosus and EAS contraction (S2, S3) with a gloved lubricated finger in the anal canal. The reflex should be elicited with repeated trials to reliably attribute EAS contraction to the glans penis or clitoral stimulation.

Certain basic laboratory studies can help to clarify the situation. A stool guaiac test is important to rule out the presence of blood; a high rectal specimen will prevent contamination from hemorrhoidal bleeding. If the person is having diarrhea without an obvious cause, *Clostridium difficile* toxin, stool culture, and examination for ova and parasites may be helpful, especially if there is a history of travel or antibiotic use in the recent past. A flat plate radiograph of the abdomen can be especially helpful to rule out impaction,[58] obstruction, or rupture of a hollow viscus. The abdominal radiograph can also be helpful in confirming and quantifying fecal retention and megacolon.[53] Liver function tests, an amylase determination, and electrolyte studies can help in the assessment of coexisting disorders.[60]

BOWEL PROGRAM: THE INDIVIDUALIZED COMPREHENSIVE BOWEL MANAGEMENT PLAN

A *bowel program* is a comprehensive individualized treatment plan focused on preventing incontinence, achieving effective and efficient colonic evacuation, and preventing the complications of neurogenic bowel dysfunction. Proper design of the bowel program includes consideration of diet, physical activity, equipment, oral medications, rectal medications, and scheduling of bowel care. *Bowel care* is the individually developed and prescribed procedure carried out by the patient or attendant to periodically evacuate stool from the colon. Time is well spent during acute rehabilitation and early community reentry in the pursuit of a long-term bowel management strategy that can be used to prevent both short- and long-term sequelae of neurogenic bowel dysfunction. Guidelines for nursing practice related to the neurogenic bowel have been developed,[61] and a panel has been convened to outline physician guidelines.[62]

Diet is an essential component of the overall program.[10] Food intake relates to the success of a bowel regimen in other important ways as well. Adequate fiber intake in the form of whole grains, fruits, and vegetables allows the stool to form sufficient bulk and plasticity to keep it flowing freely along the gastrointestinal tract.[10] The fiber component of the stool bolus retains water and maintains a plastic consistency that promotes successful propulsion through the colon. High-fat meals and dairy products tend to work in the opposite way, slowing the transit of stool.[63] Keeping the stool soft by taking in adequate fluid facilitates its transit and prevents impaction.[28] Intracolonic material in a viscous fluid phase can be propelled 32 to 100 times faster than as a solid.[22] Fluid intake targets depend somewhat on bladder status, but in general sufficient intake to produce urinary outputs of 2000 to 3000ml per day is optimal. Caffeinated beverages may have a laxative as well as a diuretic effect. Prune juice or apricot nectar can also be used to encourage elimination.

Dietary fiber is an integral part of a successful overall bowel management program. Dietary fiber includes a range of poorly digested plant-derived nonstarch polysaccharides. Soluble fiber consists of multibranched hydrophilic substances such as pectins, guar, and ispaghula which form viscous gels that delay gastric emptying and nutrient absorption as well as alleviate constipation. Insoluble fiber includes cellulose and lignin, which markedly accelerates colonic transit time in subjects with normally innervated guts. This effect is inversely proportional to basal stool weight.[64] Different types of fiber have different effects at various levels of the gastrointestinal tract. The specific effect depends on the degree of water solubility and on the location along the gastrointestinal tract. In the stomach, dietary fiber tends to prolong gastric emptying time. In the small intestine delayed absorption of nutrients may either delay or speed up transit time.[65] In the colon, fiber tends to shorten transit time. It provides bulk to stool and eases the transit along the lower bowel, and thereby increases the frequency of bowel movements. This effect is more pronounced with coarsely ground than finely ground fiber. Good sources of fiber include whole grain breads and cereals, particularly bran. Wheat germ can be added to foods to boost fiber intake if bran cereals are not palatable to the patient. Fruits and vegetables can also provide fiber as well as fluid. An added benefit of a high-fiber diet is a feeling of satiety, which may limit the intake of high-fat foods. Foods high in dietary fiber may enhance health by lowering blood cholesterol,[64,66] increasing feelings of satiety, and promoting transit of stool through the colon.[54]

The effects of dietary fiber on colonic function after SCI are not yet known. A recent investigation of the effect of dietary supplementation of SCI subjects with 40g of bran for 3 weeks revealed either no change or a prolongation of colonic transit times, with no significant change in evacuation times or in stool weight per defecation.[67] Addition of fiber to the diet of a person with SCI can successfully modify stool consistency and thus prevent hard stools and diarrhea. The expected increase in stool bulk may require an increase in the frequency and/or duration of bowel care to fully eliminate the stool in order to prevent colonic accumulation.

Following a regular schedule of bowel care is important, even if elimination of stool does not occur each time. Missed bowel care sessions can contribute to excessive buildup of stool in the colon which becomes increasingly inspissated, less plastic, and more difficult to eliminate. This can distend the colon, and thus reduce the effectiveness of peristalsis and result in extended bowel care with poor results. The

establishment of rehabilitation and spinal cord unit-wide standards for bowel management can result in uniformity of procedures, greater patient satisfaction, and improved continence of stool.[13]

Equipment plays a crucial role in the bowel care regimen. Proper positioning in order to take advantage of gravity and place the abdominal muscles at maximum mechanical advantage is necessary to facilitate the efficient passage of stool. Unfortunately, many persons have difficulty finding an appropriate commode chair.[68] A study by Nelson and associates[68] found that 37% of 147 respondents did not feel safe in their shower commode chairs. Many chairs do not have removable arm rests, and brakes were felt to be ineffective by 42% of respondents. These issues make transfers in and out of the chairs difficult. Thirty-five percent of the Nelson group's respondents reported falls during transfers, and 23% said their injuries were severe enough to require hospitalization. Development of pressure ulcers during commode chair use[69] was another issue cited, especially because the typical bowel care session was performed three times a week and took approximately 2 hours per procedure. The authors also evaluated the design of the chairs, stressing the importance of the user's being able to reach the perineal area when seated. Otherwise, the person needs to perform bowel care in bed. This may prolong the amount of time needed to complete the regimen and add to feelings of embarrassment and dependency.

MEDICATIONS: MODULATE STOOL CONSISTENCY, MOTILITY, AND DEFECATION

Medications used in the management of neurogenic bowel dysfunction can be categorized according to route of administration (oral or rectal) or pharmacologic mechanism of action. Oral medications are available in liquid or tablet form, whereas rectally administered medications are delivered as suppositories or enemas. Strategies for drug delivery to the colon are in continual development.[70] The large number of preparations available makes an exhaustive listing impractical. In addition, many medications are administered by both oral and rectal routes, may have more than one mechanism of action, or are available as combination preparations. Others may have effects that are dose-dependent, producing a mild stimulus at lower doses but leading to watery stools or diarrhea at larger doses.[71] A discussion organized by the mechanism of action of agents is expected to prepare the clinician for prescription that will meet a given patient's bowel program needs.

The major categories of agents useful to the patient with SCI include bulk-forming agents (fiber), stool softeners, laxatives (contact stimulant, saline, or osmotic), prokinetic agents, and a variety of compounded agents. Bulk-forming agents are orally administered, indigestible, nonabsorbable fibers that increase the intraluminal water content and overall volume of stool. Fiber has been shown to result in a decrease in colonic transit time in neurologically intact subjects,[65,67] probably via a reflex propulsive effect triggered by colonic wall distention due to increased fecal mass. Different types of fiber may have different effects on gastrointestinal transit times, however, and studies showing such effects involved non-spinal-cord-injured persons. As mentioned, a recent study in an SCI population suggested that increased dietary fiber consumption (in this case, bran) may actually increase overall colonic transit time.[67]

Continence is improved by modulation of stool consistency with a high-fiber diet. These agents produce uniform stool consistency by absorbing excess water to keep stool formed and by retaining water to prevent dry, hard stool. Examples include fiber from natural non-wheat sources such as psyllium (eg, Metamucil, Perdiem, Konsyl) or ispaghula husk, and synthetic compounds such as calcium polycarbophil (FiberCon) consistency and methylcellulose (Citrucel). Other beneficial effects are also observed. Psyllium has been shown to reduce serum cholesterol in hypercholesterolemic men.[66] Increased dietary fiber may also reduce the increased risk of diverticular disease observed in persons with SCI.[20,72]

Significant side effects of fiber are infrequent. Fiber supplements are generally well tolerated, and although occasional bloating or flatulence can occur, these side effects tend to resolve spontaneously.[73] These agents are usually taken with a full glass of liquid one to three times daily. Adequate additional fluid intake (generally at least eight 8-ounce glasses per day) is important with these medications to avoid stool inspissation and intestinal obstruction.[74] Serious allergic reactions to ispaghula husk can occur,[74] and this agent is not commonly used.

Fiber, whether from dietary or pharmacolgic sources, is an important component of bowel management in persons with SCI. Most of these patients need a high-fiber diet, with an intake of at least 30g of dietary fiber per day.[15] Although no definitive studies have been made to validate their use in SCI,[10] fiber supplements are believed to help prevent hard stools in patients who are unable or unwilling to consume adequate amounts of fiber in their diets.[75]

The most commonly used stool softeners are docusate sodium (Colace, Surfak) and potassium (Dialose). Their proposed mechanism is to lower the surface tension of stool and thereby allow water and lipids to enter and soften the fecal mass, although stool volumes increase negligibly with docusate sodium.[76] They may also have an irritating action on the mucosa by stimulating the secretion of mucus, water, and electrolytes.[77] Although docusate itself does not directly increase colonic motility and has no laxative action, it has been demonstrated to increase stool frequency in an elderly population.[71,73,78] Long-term use in persons with SCI must be carefully considered, preferably with objective assessment of the therapeutic effects experienced by individual patients. Docusate may increase the uptake of other drugs,[71,74] and thus increase their potential toxicity. Its oral use is best reserved for situations in which straining must be avoided, as in patients with painful hemorrhoids,[73] autonomic dysreflexia, or angina.

Other agents with stool-softening action include liquid paraffin, mineral oil, and seed oils such as croton and arachis oils. They are also described as intestinal lubricant laxatives.

These drugs may interfere with the absorption of fat-soluble vitamins. Mineral oil has been reported to cause lipid pneumonia following aspiration. Because of these undesirable effects, the use of such agents has been generally discouraged.

Stimulant laxatives act by enhancing intestinal motility and thereby decreasing the time available for water and electrolyte resorption. These agents include anthraquinone-containing substances obtained from senna (Senokot), cascara, aloe, or rhubarb, and chemically similar synthetic drugs. Senna is a glycoside that is split by colonic bacteria into absorbable anthraquinones.[73] It generates increased propulsive activity by altering electrolyte transport and increasing intraluminal fluid. It also exerts a direct stimulant effect on the myenteric plexus which increases intestinal motility.[79] Oral senna agents are widely used among persons with SCI. They work best in UMN-level injuries, and facilitate bowel movements in 6 to 12 hours. Thus, for an evening bowel care session they should be taken early in the day, and vice versa.[63]

Polyphenolic derivatives represent another group of commonly used stimulants. This group includes bisacodyl (Dulcolax) and phenolphthalein (Ex-Lax and others). Bisacodyl is found in many common oral and rectal agents. Orally, its usage pattern is similar to that of senna. Castor oil is another agent in the stimulant category and was once in common use among the general population. It is now of historical interest only, as it is seldom prescribed because of side effects of cramping, watery bowel movements, malabsorption, and dehydration.[71,73]

All stimulants share, to some degree, the potential for dose-dependent side effects, including abdominal cramping, diarrhea, and electrolyte imbalance. The senna preparations, bisacodyl, and phenolphthalein are the most commonly used agents. Phenolphthalein is a parent compound which is structurally related to, and led to the discovery of, bisacodyl. It is still in use in many over-the-counter cathartics. Adverse effects include dehydration and electrolyte imbalance, and thus its use should be avoided in elderly patients.[73] Bisacodyl suppositories are generally better tolerated. In the SCI population, they are used early after SCI with each bowel care session to help establish an effective bowel program.[15] They are also used on a long-term basis by some. However, they may cause mucosal irritation[80] and therefore may not be appropriate for daily use in some patients. Ideally, many patients can be weaned from suppository use and taught to rely on oral medications and digital stimulation,[15] but lack of the person's hand function and preferences of the attendant may make this impractical.

Bisacodyl comes in many common orally and rectally administered nonprescription preparations. In suppository form, it is the most commonly used rectal stimulant. It is available in formulations with either a hydrogenated vegetable oil base or a polyethylene glycol base (ie, the "Magic Bullet"). There is evidence that a polyethylene glycol-based bisacodyl suppository can produce significantly more rapid onset of defecation in patients with SCI and shorten the total bowel care time.[81-83]

Two conditions, "cathartic colon"[73] and melanosis coli,[74] have been reported in association with chronic use of stimulant laxatives, most notably with the anthraquinone derivatives such as senna, aloe, and cascara. Clinically, cathartic colon presents with a progressive decrease in responsiveness to irritant laxatives over time. However, the incidence and pathophysiologic mechanism of this process remain to be elucidated.[73,74] Some authors have suggested that neuropathic damage to the myenteric plexus is responsible,[84] but studies are still inconclusive.[73,74] Melanosis coli is a colonoscopic and histologic finding that appears as staining of the colonic mucosa and results from macrophage phagocytosis of pigments derived from gut stimulants. No association with specific symptoms has yet been made,[73,74] although it has been suggested that a dilated, atonic colon could occur.[10]

Saline laxatives are salts, generally of magnesium, sodium, or potassium. By far the most commonly used agent of this type is over-the-counter oral magnesium hydroxide (Milk of Magnesia). Other common agents include magnesium citrate solution (used orally) and sodium phosphate/biphosphate (Fleet's Phosphosoda oral solution or enema). The oral preparations act by drawing fluid into the small intestine, where it induces mixing actions and stimulates colonic motility. Magnesium hydroxide also causes release of cholecystokinin, which may further stimulate motility.[85]

Saline enemas act directly on colonic mucosa in a similar fashion, causing an influx of water and electrolytes, which stimulate the distal colon and rectum. These agents share a rapid onset of action (evacuation usually occurs within 2 to 6 hours). However, they often provide a rather potent, unpredictable stimulus, with resultant abdominal cramping and watery bowel movements.

These agents (excluding Milk of Magnesia) are most useful when taken in large volume to induce complete bowel evacuation. Other agents with more gentle stimulant effects are generally preferred for establishing a bowel routine in patients with SCI. In addition, significant side effects may be associated with particular preparations, including dehydration, hypermagnesemia,[73,74] hyperkalemia, and congestive heart failure.[74] Enema usage should be restricted to those times when the bowel care procedure becomes ineffective despite use of suppositories and digital stimulation,[86] for example if there is a lack of a bowel movement for 3 or more days.[63] Long-term usage can result in an enema-dependent bowel, and may deplete important nutrients from the bowel.[63] Enemas are associated with many potential complications: rectoanal trauma, bowel perforation, electrolyte disturbances, bacteremia, colonic infections, and autonomic dysreflexia.[87]

Hyperosmolar laxatives include lactulose, sorbitol, and polyethylene glycol electrolyte solutions (Colyte, GoLytely). These agents are metabolized in the colon into short-chain amino acids, which act osmotically to draw fluid intraluminally. Lactose and sorbitol have generally mild effects on motility, while the polyethylene glycol electrolyte solutions act more rapidly with stronger bowel evacuation effect. Polyethylene glycol electrolyte preparations are commonly used to cleanse the bowel prior to operative or endoscopic procedures. They have two advantages for this purpose: they do not cause either electrolyte imbalance or mucosal irritation.

Lactulose and sorbitol, on the other hand, cause cramping and flatulence, which may be self-limited[74] or persistent.[71]

Glycerine is generally used in suppository form and stimulates rectal contraction through hyperosmotic and irritant actions;[71] it generally produces a bowel movement in 15 to 30 minutes. Glycerine suppositories are often used during the transition from bisacodyl suppositories to digital stimulation in patients with SCI because they provide a less potent chemical stimulus.[15,86] Patients can be put on a schedule of alternating bisacodyl and glycercine suppositories. The frequency of digital stimulation may need to be increased with the glycerine suppositories in order to achieve similar results. In many patients, transitioning to glycerine as the sole chemical triggering agent is possible, alternating with just digital stimulation. Thereafter, similar results can be acheived with digital stimulation alone used to trigger and maintain the progress of defecation.

Prokinetic agents act chemically by affecting neurotransmitter levels to stimulate gastrointestinal motility. Two such drugs are currently in use in the United States: metoclopramide (Reglan) and a newer medication, cisapride (Propulsid).

Metoclopramide increases gastric motility, with no effect on the colon.[88] Its exact mechanism of action is unclear, but it appears to sensitize tissues to acetylcholine. It has been used effectively to promote gastric emptying[89] and to resolve ileus after acute SCI.[90] It also exerts an antiemetic effect, probably via antidopaminergic action. Metoclopramide can cause several dose- and duration-related adverse reactions, including dystonic reactions (which are occasionally irreversible) and other extrapyramidal side effects.

Cisapride, which has come into increasing use in the past few years, increases motility throughout the gastrointestinal tract. This agent works locally by facilitating the release of acetylcholine from the postganglionic cholinergic nerve endings at the myenteric plexus.[91] It was originally released for treatment of chronic refractory constipation. Case reports suggested its clinical utility for patients with intractable constipation.[92,93] Preliminary studies have demonstrated that it normalizes mouth-to-cecum transit times,[92] left colon transit times,[94] the abnormally long mouth-to-cecum transit times in quadriplegics,[47] and mouth-to-anus transit times.[95] Other observed effects include reduced urinary retention,[92,93] increased rectal tone,[95,96] and a subjective reduction in defecation time.[95] It may be useful in refractory constipation in patients with SCI. It was believed to have few side effects[74] until recently, when an association between its use and the cardiac arrhythmia torsades de pointes was reported.[97]

Several agents are available that are used more commonly among patients with SCI than in the general population. Some, in fact, were developed to meet their unique needs. Examples include Therevac mini-enemas and CO_2 suppositories. Therevac mini-enemas are provided in a liquid form and are so named because they contain only a small volume of fluid (4ml). They are composed primarily of docusate sodium in a soap base of glycerin and polyethylene glycol. A variety is also available with benzocaine, which anesthetizes the rectal mucosa. This can be useful for those patients with SCI who experience autonomic dysreflexia as a result of nociceptive stimuli from performance of their bowel care. Therevac mini-enemas likely trigger reflex-mediated colonic peristalsis by acting as a mucosal stimulus, and they provide lubrication by penetrating stool and softening it by the action of docusate sodium. They have a rapid onset of action, generally producing a bowel movement within 15 minutes.[82,98,99] When compared with polyethylene glycol-based bisacodyl suppositories, they initiate defecation within similar time periods and produce comparable amounts of stool.[82]

CO_2 suppositories (eg, Ceo-Two) act by producing carbon dioxide gas, which causes rectal distention and a reflex increase in colonic peristaltic activity. A mechanical expulsion of stool is probably produced as well. However, these suppositories may not produce predictable results, because of premature leakage of the carbon dioxide past the paralytic anal sphincters of persons with SCI.[3] They may be more effective in SCI patients with low-level injuries[85] who lack reflex defecation but can obtain elimination through mechanical effects.

BOWEL CARE: THE PROCEDURE FOR FACILITATED DEFECATION

Normal defecation can be operationally defined as the easy volitional passage of enough soft formed stool on a regular basis to prevent the sensation of incomplete evacuation. After SCI, the goal is to reachieve as normal and efficient a defecation task (bowel care) as possible. This is done with an individually developed plan for elimination of stool carried out as scheduled bowel care. The goals of bowel care are to facilitate controllable defecation of the maximal stool volume in the least amount of time, with avoidance of stool incontinence thereafter. Without the ability to feel stool in the rectum or to consciously initiate reflex defecation, a person with SCI must regularly assume the need for bowel movements if he or she is to predictably eliminate stool and avoid colonic overdistention. Therefore, bowel care is a scheduled process of facilitated reflex defecation. Initially, during the acute period of spinal shock, there is a reduction in peristalsis, requiring digital or manual removal of stool.[100] After colonic function stabilizes, one of two basic procedures of bowel care is utilized. These techniques use residual motor function after UMN and LMN injuries in order to meet the goals of the bowel care.

Persons with SCI who have LMN injuries often have more difficulty with their bowel care because of the absence of spinal reflex peristalsis (areflexic bowel) and EAS tone. The rectum must be cleared of stool more frequently, usually one or more times per day, to prevent unplanned defecation of stool that cannot be retained by the patulous external sphincter.[3,46] Patients should be taught to avoid Valsalva forces during transfers to prevent expulsion of stool. Some patients wear tight underwear or bicycle pants to support the pelvic floor and help retain stool. When these are used care should be taken to prevent skin breakdown by checking for skin tolerance along seams. An air or gel cushion is also helpful to evenly distribute pressure across the descending perineum.

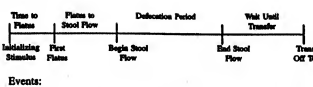

Fig 4. Events and intervals of bowel care. Bowel care events separate the total period into discrete intervals. Pharmacologically or mechanically initiated bowel care begins with insertion of rectal medication or digital-rectal stimulation which acts as an *initializing stimulus*. *First Flatus* ends the interval *Time to Flatus* (initializing stimulus until first gas is passed). *Begin Stool Flow* ends the second interval, termed *Flatus to Stool Flow*, and begins the *Defecation Period*. *End Stool Flow* represents the time when defecation has immediately ceased. The time *Transfer off Toilet* ends the *Wait Until Transfer* period, which represents the time spent to insure that defecation is complete. The time of *Transfer Off Toilet* ends the bowel care procedure.

The LMN bowel care procedure usually consists of removing stool with the finger and may include digital stimulation to enhance local segmental peristalsis. Bowel care starts in the seated position on the commode. The gloved and lubricated finger is introduced into the rectum, and the internal sphincter is relaxed by circular finger movement. Intermittent Valsalva force and abdominal wall contraction along with transabdominal massage of the colon in a clockwise manner helps advance the stool. Stool is hooked with the finger in the rectum and digitally evacuated. Digital removal is repeated with digital stimulation until there is no further palpable stool. The absence of palpable stool and/or tightening of the IAS signals the end of bowel care.

After complete SCI above the level of the conus medullaris, there is no conscious control of the external anal sphincter, a limitation in the production of effective intra-abdominal pressure, and a lack of discriminate rectal sensation. However, many subjects with a clinically complete UMN bowel are able to perceive a nonspecific deep pelvic sensation when a balloon catheter distends the rectum.[52] The UMN bowel care procedure must overcome the impairments of infrequent propulsive peristalsis, spastic EAS, and deficits of rectal sensation.

Persons with UMN injuries need to anticipate the urge for defecation which they may not feel with a scheduled trigger of defecation every 1 to 3 days. They stimulate the reflex manually with a finger (or assistive device) inserted in the rectum (digital stimulation) with or without an appropriate chemical or electrical stimulus. The initial trigger for defecation is typically a suppository, enema, or mini-enema, which produces a mucosal contact stimulus that initiates conus-mediated reflex peristalsis. The chemical stimulant is placed directly against the mucosa in the upper rectum. A waiting period (fig. 4) then starts as the active ingredients dissolve, disperse, and stimulate. Stool flow then begins and is augmented as necessary with digital stimulations.

There are many methods for initiating defecation in persons with UMN (reflexic) bowels. Historically, enemas were used as a mechanical stimulus to trigger defecation, soften stool, and cleanse the colon after SCI.[101,102] In 1987, Shandling and Gilmore[103] reported 100% success with use of the enema continence catheter in the management of 112 children with spina bifida. Liptak and Revell [87] used a modified retention catheter with a study population of 31 children with meningomyelocele. The bowel care frequency was once every 1 to 2 days. They achieved an increase in continent stools from 28% to 94% ($p < .01$). Pulsed irrigation-enhanced evacuation is a programmed enema delivered in a series of tap water pulses that rehydrate, suspend, and drain away the stool.[104] The enema is delivered through a tube to a rectal speculum with an inflated retention cuff and a conduit to drain effluent with stool to a reservoir. Devices that deliver pulsed irrigation-enhanced evacuation have been successfully used to clear impactions as well as for regular bowel care of persons with an LMN or a UMN bowel. An alternative method of enema delivery is through a surgically created continent catheterizable appendicocecostomy stoma.[105] Tap water or a mild soap solution is infused directly into the cecum through the right abdominal stoma in volumes of 300 to 800ml, inducing colonic evacuation in 8 to 32 minutes.[106]

Functional electrical stimulation of defecation has been under investigation intermittently for decades.[107,108] Recently, urination[108] and defecation have been triggered with electrical stimuli by some investigators with selective nerve root activation in patients with a UMN bowel.[108-110] Electrodefecation with neuroprosthetic stimulation is achieved by radio frequency activation of a subcutaneous receiver that stimulates the S2, S3, and S4 nerve roots bilaterally.[42] Trains of stimuli at low or high frequency are delivered for up to 10 seconds, then repeated. Complex high-pressure phasic colonic and rectal contractions, resembling peristaltic activity, are produced. Defecation ensues after the train of stimuli. S4 nerve root stimulation produced anal sphincter contraction.[42,111]

If the SCI lesion is above the conus medullaris, the recto-colic reflex can be exploited in bowel care. *Digital stimulation* is a technique for inducing a reflex peristaltic wave from the colon to evacuate stool. This maneuver is accomplished by gentle insertion of the entire lubricated gloved finger into the rectum. Allergy to latex is common and should be considered in the choice of gloves used.[112] After the stimulation finger is fully inserted, gentle sustained pressure toward the sacrum relaxes the puborectalis muscle, and thereby makes the rectal angle less acute and reduces outflow resistance. This also opens the EAS and provides a stretch stimulus that further reduces spastic tone and rectal outflow resistance. Rapid excessive stretching or noxious stimuli should be avoided, as they can precipitate anal sphincter spasm. Stimulation is delivered by a gentle rotation in a circular manner, dilating the distal rectum and anal canal. It is important to continually maintain contact with the mucosa. Rotation is continued until relaxation of the bowel wall is felt, flatus passes, stool comes down, or the internal sphincter constricts. This maneuver activates the rectoanal inhibitory reflex, producing relaxation of the internal sphincter, and the recto-colic reflex, stimulating pelvic nerve-mediated peristalsis and also promoting local enteric-coordinated peristalsis. Rectal contractions in response to rectal distention are

stronger in persons with UMN SCI.[49,52] Section of the posterior roots eliminates the rectal contractile response to rectal distention, and confirms that it is spinal reflex mediated.[52] In practice, digital stimulation longer than 1 minute at a time is seldom necessary. Peristaltic activity and stool delivery in response to a digital stimulation occurs within seconds to a few minutes. During bowel care, digital stimulations are repeated every 10 minutes as indicated to maintain progress with defecation.

The end of bowel care is signaled by cessation of flatus and stool flow, palpable internal sphincter closure, or the absence of stool results from the last two digital stimulations. Patients frequently "sense" the end of the bowel movement. This perception is possibly mediated by visceral afferents transmitted through the sympathetic chain, or partial sacral sparing of anal afferents relayed through the spinal cord.

Many persons with SCI report that various techniques enhance their bowel care. Increased activity may help; some do range-of-motion exercises before bowel care. Some persons with UMN SCI report stimulating defecation with cutaneous stimulation of sacral dermatomes. Transabdominal massage starting in the right lower quadrant and following the course of the colon in an aboral direction has been used in an attempt to enhance propulsive peristalsis and bring stool to the rectum.

Although a popular form of alternative medicine, abdominal wall massage has not proven effective in constipated or asymptomatic subjects without SCI.[113] Abdominal wall massage along the course of the colon has been included by many with SCI as a prelude and an adjunct to bowel care for years.[102] The efficacy of massage is yet to be tested in SCI populations. However, mechanical stimulation of the rectum produces supranormal rectal and sigmoid contractions in UMN chronic SCI.[49,52] Transabdominal massage could conceivably trigger promulgated peristalsis as well.

COMPLICATIONS FROM NEUROGENIC BOWEL DYSFUNCTION: EFFECTS OF TREATMENT AND AGING

For a person with SCI, neurologic impairments contribute to colonic pathophysiology as well as to dysfunction affecting many other organ systems. As a result, there is potential for adverse interactions from treatment of other SCI-related conditions which could affect colonic function. Moreover, the prolonged effects of neurogenic colonic dysfunction in addition to the effects of aging are only begining to be understood.[114]

Medications can complicate colonic function in a variety of ways. Anticholinergic medications can slow gut motility or dry the stools to the point that flow along the gut is affected. Narcotics, particularly in the acute setting, can affect elimination adversely. It is not uncommon for a patient with SCI who has recently undergone surgery to experience ileus and fecal impaction.[58] In such cases, anesthetics, narcotics, and the effects of injury all potentially play a role.

Broad-spectrum antibiotics can cause diarrhea by disrupting the ecology of symbiotic resident colonic microbes. Such effects can be mitigated with the use of *Lactobacillus* capsules (eg, Lactinex) or the consumption of yogurt made with live *Lactobacillus* cultures in an attempt to reestablish a more favorable colonic flora. These organisms have been called "probiotics" because they produce a healthy balance of colonic microbiota.[27] Dietary modulation to include "prebiotics," defined as foodstuffs promoting the growth of desirable colonic microorganisms (eg, bifidobacteria and lactobacilli), has also been proposed. Inulin and oligofructose, which are contained in foods such as garlic, onion, artichokes, and asparagus, are representative prebiotics.

Diarrhea can be related to gastrointestinal infection, food intolerance, or use of antibiotics (most commonly to treat urinary tract infection). Treatment in those cases is similar to that for patients without neurogenic bowel, namely using antidiarrheal agents, doing workup for infectious causes, including *C. difficile*, when indicated, and discontinuing use of offending agents. Commonly, diarrhea alternating with constipation can be seen, which is often related to partial bowel obstruction with flow of diarrheal stool around an impaction.[58,115] The paradoxic cause of impaction with diarrhea has been attributed to the ball-valve effect of an intermittently obstructing fecal mass.[116] This can be observed as stool-filled loops of bowel on plain radiographs and requires complete evacuation of the bowel with oral magnesium citrate preparations, polyethylene glycol electrolyte solutions, or enemas with pulsed irrigation-enhanced evacuation. *C. difficile* colitis should be a consideration in SCI patients who have had antibiotics 6 or more days before the appearance of loose stools. A Wright-stained smear revealing fecal leukocytes suggests more severe colonic inflammation and is a simple adjunct to testing for *C. difficile* toxin. When testing for *C. difficile* toxin, the positive predictive value reaches 91% and the negative predictive value after two specimens with a negative result is 97%.[117] The cause of diarrhea must be carefully defined in order to provide the most efficacious treatment. For example, the use of antidiarrheal agents in the setting of impaction can exacerbate the condition.

Long-term use of stimulant laxatives containing anthraquinones, such as senna, aloe, and cascara preparations, should be avoided because they may produce neuropathic damage to the myenteric plexus.[118] Pigments from such laxatives can stain the colonic mucosa and produce melanosis coli. Osmotic agents are preferred for ongoing use because neither organ damage nor tolerance develops. Daily fiber supplements containing cellulose, polysaccharide, or psyllium can improve stool consistency if adequate fluid intake is maintained. Long-term suppository use is not known to cause colonic complications. Attempts can be made over time to wean patients from suppositories and have them rely solely on reflex emptying from digital stimulation.[15,98]

Gastrointestinal complications requiring surgical attention are quite frequent after SCI. The gastrointestinal tract can contribute to abdominal emergencies, which can produce as many as 10% of deaths after SCI.[119] Sensory deficits after SCI significantly delay the diagnosis. Many of these conditions originate from the colon, appendix, rectum, or anus,[20,120] including appendicitis, sigmoid volvulus,[121] diverticulitis, ischemic colitis, obstruction, and impaction.[58,120]

Proximal colonic impactions often arise in patients with a UMN bowel, whereas distal impactions are seen in those with an LMN bowel.[20] This pattern is attributed to the loss of spinal reflex-mediated distal colonic peristalsis after LMN SCI. The presenting symptoms of acute abdomen in SCI are frequently nonspecific, such as abdominal distention, vomiting, and constipation. Early diagnosis requires clinical suspicion, laboratory evaluation (complete blood count, amylase), and appropriate imaging studies (abdominal radiograph, CT of abdomen).

Bowel care may exacerbate a number of conditions. Persons with SCI who are at risk for autonomic dysreflexia can become symptomatic with rectal stimulation or colonic distention from carbon dioxide–producing suppositories. Use of milder mucosal contact stimulants, topical anesthetic lubricants, and premedication with nifedipine before bowel care is sometimes necessary.

Hemorrhoids can be managed by minimizing trauma and avoiding unsupported seating. Seating can be designed to maintain pressure support for the perineum between bowel care sessions. Supporting the pelvic floor with a gel or air cushion to distribute pressure over the entire perineal surface may help prevent the enlargement of hemorrhoids and help maintain closure of the anal sphincter. Dietary prevention strategies include a high-fiber diet to maintain a soft plastic stool that is propelled without excessive intracolonic pressures. Should hemorrhoids become a frequent source of bleeding or autonomic dysreflexia, banding, an outpatient procedure, has offered relief to persons with SCI.[122]

Spinal cord injury can contribute to the early development of diverticuli.[20] The causes for this have not yet been conclusively identified. Myoelectric studies of the UMN colon have suggested that there is excessive colonic activity as compared with controls without SCI.[39] Other investigators have recorded high intracolonic pressures on colometrograms in persons with UMN SCI.[51] In neurally intact populations, physical activity and high fiber content of the diet are associated with a decreased incidence of diverticuli.[72]

Surgical intervention for colonic dysfunction is carried out in selected populations for extreme constipation[123] and after SCI as adjunctive treatment for pressure sores [124] or for extremely long bowel care time.[14] Pressure ulcers alone may not be the best indication, as healing is not always accomplished with a single intervention of fecal diversion through a colostomy.[125] Patients must be good operative candidates and report significant difficulty or complications with typical bowel care as well as demonstrate the capacity to manage and benefit from a colostomy.[126] Surgical complications are significant. The risk of death as part of the postoperative course after colostomy with concurrent large pressure sores has been reported to be as high as 15% in a small series.[125] Once in place, colostomies are generally well received.[126] Patients note reduced time requirements for bowel care and enhanced quality of life.[124-127] Consideration of fecal diversion should therefore include a broad assessment of bowel program, bowel care, personal assistance, body image, and lifestyle issues. Once the decision for fecal diversion is made, the surgical procedure and the location of the stoma require interdisciplinary assessment, including primary rehabilitation nursing and occupational therapy. In our experience, high placement of the stoma allows the best patient visualization and self-management.

Colorectal carcinoma has been associated with aging, Western society, familial syndromes, ulcerative colitis, and ureterosigmoidostomy. Myelopathy may increase the risk by producing constipation and increasing the duration of stool storage. The multiple nonspecific gastrointestinal symptoms, sensory deficits, and colonic impairments of persons with SCI can make colorectal disease an occult diagnosis.[120] The incidence of colorectal carcinoma has been estimated to be two to six times greater among persons with SCI,[127] although a population-based study of veterans with SCI revealed tumor distribution and stages similar to those expected with neurally intact patients.[128] The added risk for colon cancer which comes with myelopathy, if any, needs to be further elucidated. In the interim, the recommended screening should be carried out in the SCI patient population. Digital rectal examination has been recommended annually for asymptomatic persons 40 years of age or older and from age 35 for persons with first-degree relatives with a history of polyps or cancer. Persons at normal risk should be screened with sigmoidoscopy or colonoscopy every 3 to 5 years beginning at 50 years of age.[129] Flexible endoscopy screening with polypectomy as a preventive measure has been estimated to reduce the risk of rectal and colon cancer up to 50% in a general veteran population.[130] Hemorrhoidal bleeding can complicate stool guaiac screening with false-positive results, and colon cleansing for examination can be complicated by electrolyte disturbances and orthostasis.

CONCLUSIONS: REHABILITATION RESEARCH TO IMPROVE BOWEL MANAGEMENT AND QUALITY OF LIFE

The neurogenic colon remains a bane to the existence of persons who have SCI. Rehabilitation interventions have changed little over the past 50 years. Advances within the fields of gastroenterology and proctology[131] bring alternatives that may offer persons with SCI improved bowel programs in the future. The colon represents a fertile frontier for investigation and innovation to improve quality of life after SCI.

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1997 SAE-P: Neurogenic Bowel Focused Review

1. Fecal continence in the resting state is primarily maintained by the tonic activity of which of the following muscles?
   (a) Internal anal sphincter
   (b) External anal sphincter
   (c) Puborectalis
   (d) Pubococcygeus
   (e) Levator ani

Ref: Schweiger M. Method for determining individual contributions of voluntary and involuntary anal sphincters to resting tone. Dis Colon Rectum 1979; 22:415-6.

2. Parasympathetic innervation of the descending colon is provided by
   (a) the vagus nerve
   (b) the pelvic nerve (nervi erigentes)
   (c) mesenteric nerves
   (d) hypogastric nerves
   (e) pudendal nerves

Ref: Sarna SK. Physiology and pathophysiology of colonic motor activity, part I. Dig Dis Sci 1991; 36:827-62.

3. You are asked to evaluate a 32-year-old patient with C6 motor and sensory complete tetraplegia who complains of alternating bouts of diarrhea and constipation with abdominal distention over the last 2 months. There has been no change in medications or diet. The most likely diagnosis is
   (a) colonic infection
   (b) partial bowel obstruction
   (c) autonomic dysreflexia
   (d) ulcerative colitis
   (e) mesenteric artery syndrome

Ref: (a) Juler GL, Eltorai IM. The acute abdomen in spinal cord injury patients. Paraplegia 1985; 23:118-23. (b) Wrenn K. Fecal impaction. New Engl J Med 1989; 321:658-62.

4. Which of the following is a complication that could result from the use of digital-rectal stimulation as part of bowel care for a patient with C6 tetraplegia?
   (a) Incontinence of mucus 1 hour after bowel care
   (b) Autonomic dysreflexia
   (c) Mucosal laceration
   (d) Melanosis coli
   (e) Laxative-dependent colon

Ref: Stone JM, Nino-Murcia M, Wolfe VA, Perkash I. Chronic gastrointestinal problems in spinal cord injury patients: a prospective analysis. Am J Gastroenterol 1990; 85:1114-9.

5. You are asked to begin a bowel program on a spinal cord-injured patient with recent onset of a complete lower motor neuron lesion. You recommend the following bowel care procedure:
   (a) stimulant laxatives every other day
   (b) saline enemas
   (c) bowel care to be carried out twice weekly
   (d) use of Valsalva maneuver
   (e) digital-rectal stimulation with manual removal of stool

Ref: Stiens SA, Goetz LL. Neurogenic bowel dysfunction. In: O'Young B, Young MA, Stiens SA, editors. Physical medicine and rehabilitation secrets. Philadelphia: Hanley & Belfus, 1997:460-4.

1997 SAE-P: Answer Key and Commentary on Preferred Choice: Neurogenic Bowel Focused Review

QUESTION	ANSWER	COMMENTARY
1.	(a)	The internal anal sphincter remains closed in the resting state to provide the initial barrier to fecal incontinence. Should there be an unanticipated surge of stool into the rectum, reflex contraction of the puborectalis will occlude the rectal lumen by making the rectal angle more acute and the external anal sphincter will contract. The levator ani raise the pelvic floor during defecation.
2.	(b)	The pelvic nerve (S2-4) is the parasympathetic innervation to the descending sigmoid colon and rectum. The vagus nerve provides the parasympathetic innervation to the entire alimentary canal up to the transverse colon. The mesenteric nerves provide sympathetic innervation to the colon, and the pudendal nerves provide the innervation to the external anal sphincter.
3.	(b)	Partial bowel obstruction occurs frequently after SCI and is most often located in the colon. Fecal impaction is notorious for this presentation, which results from the "ball valve effect" of an obstructing fecalith. Colonic infection and ulcerative colitis would be less likely to cause intermittent constipation and would be associated with a persistent pattern of diarrhea. Superior mesenteric artery syndrome presents with vomiting and abdominal bleeding. An initial laboratory evaluation in this case might include an abdominal radiograph, complete blood count, serum amylase, and stool examination for occult blood, fecal leukocytes, and *Clostridium difficile* toxin.
4.	(b)	Autonomic dysreflexia (AD) can occur in response to any noxious stimulus below the spinal cord injury level. If AD occurs in response to digital-rectal stimulation, 1% lidocaine jelly can be used to minimize the pain stimulus. Incontinence of mucus after bowel care has ended is an occasional occurence that follows the use of a chemical stimulant to initialize defecation. Use of a less potent chemical stimulus or a smaller dose frequently solves the problem. Mucosal laceration is uncommon if adequate lubrication is used, although some anal rectal bleeding with bowel care is reported by up to 30% of persons with SCI. Melanosis coli is an endoscopic finding of mucosal pigmentation from the chronic use of some laxatives. A "laxative dependent" colon can develop with chronic use of oral stimulant laxatives.
5.	(e)	Digital-rectal stimulation opens the internal anal sphincter and dilation of the rectum stimulates local peristalsis via interactions between the submucosa and myenteric plexus. Digital hooking of the stool evacuates the rectum. Stimulant laxatives may evoke an unanticipated defecation or liquid stool. Saline enemas may leave behind residual fluid that could contribute to incontinence when bowel care is over. Bowel care should be carried out daily or more frequently in persons with lower motor neuron SCI, because stool that is advanced into the rectum by giant mass contractions can be inadvertently released past the internal anal sphincter. Although the Valsalva maneuver can help expel stool, it does not reliably move stool beyond the internal anal sphincter and therefore remains an adjunct to bowel care and an activity to be avoided in the interim.